corneoscleral perforating injury of the left eye was noted, with iris, ciliary body and vitreous prolapse, total hyphaema and loss of the crystalline lens. The eyelids were intact. Abscision of damaged uvea, anterior vitrectomy and wound repair was performed. The scalp and facial lacerations were explored, cleaned and repaired in layers. Postoperatively an anterior vitreous membrane prevented visual improvement beyond 6/36 and anterior vitrectomy was performed at 7 weeks postinjury. The eye now sees 6/5 with a contact lens.

Case 2: A 56-year-old man was attacked by his own dog, a Pit Bull Terrier. The patient was reticent as to the exact circumstances of the injury.

On presentation 24 h later, there was a puncture wound over the right temple, and a complex perforation of the right globe, comprising a circumferential medial scleral rupture, and a corneal laceration crossing the visual axis. The crystalline lens was disrupted but present. The anterior segment was cleared of vitreous and visible lens matter and the wounds repaired. Persistent hyphaema, and fibrinous uveitis with retained lens matter made further surgery necessary within 7 days. Anterior segment clearance revealed cyclodialysis and retinal detachment, necessitating globe encirclement and gas tamponade. Postoperatively a total retinal detachment supervened and the patient refused further surgery. The eye is blind.

Discussion

Even in the presence of severe periocular lacerations caused by dog bites, the eye is usually undamaged[4,5]. Occasionally, however, the orbit is entered, and lacrimal gland injury[6], transsection of the medial rectus[7] and trochlear damage[8] have been reported. Perforating injury itself is rare. It seems likely from the location of these injuries that the upper canines provided a fulcrum on the cranium while a lower canine entered the orbit inferomedially with sufficient force to perforate the globe.

These two patients demonstrate contrasting visual outcomes. The reasons for this are numerous, but in case 2, the late presentation, more extensive ocular damage, retained lens matter and necessity for surgical re-intervention within the first week are most important. In view of the perceived risks of wound infection[2-5], it is notable that in neither case did bacterial endophthalmitis develop.

A previous survey[9] identified the German Shepherd as the dog breed most frequently responsible for facial injuries. However, others are also perceived as high-risk, and both the above breeds would appear in this group. Further documentation is necessary.

Injuries of this severity caused by dog attacks are distressing for patient and relatives, and children are usually the victims. Palmer[1] found that 87% were under 15 years of age. The subsequent destruction of the animal merely provides a scapegoat. Clearly situations which entail a high risk of injury should be avoided. This implies improved control of dogs by their owners, especially in the presence of children, and legislation to this end may prove necessary.

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Acute Guillain-Barré syndrome presenting as acute spinal cord compression in an elderly woman

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Keywords: Guillain-Barré syndrome; spinal cord compression

Guillain-Barré syndrome (GBS) is a polyneuropathy believed to be mediated by an autoimmune mechanism[1]. Peak incidence is bimodal (15-34 years and 50-74 years) but diagnosis is rare beyond 75 years[2]. We describe severe acute GBS in an elderly woman who required intensive care within 48 h of hospital admission.

Case report

An 81-year-old widow with a previous history of spinal osteoporosis and vertebral collapse of D12/L1 was mobile, living independently and was on no regular medication. She retired to bed feeling well, but on attempted rising the following morning felt severe pain in both thighs and could not move her legs. She was referred urgently with diagnosis of acute spinal cord compression.

On admission she complained of diffuse numbness and weakness in the legs. There was generalized hypotonia, particularly in the legs, with areflexia. There was a flicker of movement at the toes and grade 3 muscle power in the arms. Gag reflexes were intact and no ophthalmoplegia was noted. Sensation was absent below the umbilicus. She was continent of urine. She was noted to have dysphonia and admitted to some dysphagia but there were initially no respiratory difficulties and clinical assessment was otherwise unremarkable.

The following investigations were normal; erythrocyte sedimentation rate, full blood picture, blood glucose and electrolytes. The cerebrospinal fluid (CSF) protein was 0.7 g/l. Within 48 h the patient was aphonic with increasing dysphagia, reduced gag reflexes and increasing weakness of the neck muscles. She developed respiratory distress and was unable to maintain normal blood gas levels breathing 60% oxygen. She was then transferred to intensive care where she was intubated and ventilated. Limited plasmapheresis was performed over the next 5 days. Repeat CSF protein level was 0.9 g/l. Autoimmune screen, viral antibody titres for rubella, adenovirus, mumps, measles, herpes and varicella, and antibody titres for the psittacosis, Q-fever and mycoplasma organisms were negative in serum. The CSF antibody titre levels were negative and thyroid function, B_{12} and folate levels were normal.

During the next week upper limb muscle power deteriorated to grade 2. Four weeks later respiratory function improved and ventilation was discontinued for most of each day. Despite requiring aggressive intravenous antibiotic and inotropic therapy for septicaemic shock associated with pneumonia, muscle power continued to improve and after a further 4 weeks she was returned to general ward care with a tracheostomy. This was allowed to close off. Ventilation is now satisfactory and speech, swallowing, and gag reflexes are normal. Muscle power at the neck, shoulders

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and elbows is normal. Power at the hips, knees, and wrists is grade 3 and at the ankles remains zero. Tendon reflexes have returned in the arms and knees and sensation has returned fully. Fine hand function remains impaired and the patient can walk a few steps with support.

Discussion

Guillain-Barré syndrome is often thought to have a benign prognosis, but 7% of patients die and a further 16% suffer residual disability[3]. Early diagnosis and management may shorten the course and reduce morbidity[1]. Plasma exchange within 7 days of onset of neurological symptoms reduces ventilation time and hastens clinical improvement[1].

Features of patients with poor outcome include severe deficit requiring ventilation and failure to improve within 3 weeks of reaching peak deficit[3]. Quadriplegia appearing over 2-5 days is associated with the most severe residual deficit leading to permanent chairbound status[4]. However, an unduly pessimistic attitude towards recovery for the severely disabled at 6 months does not seem justified, since improvement may only plateau at 18-24 months[4].

In the elderly, prospects for recovery are quite good[2]. This woman's illness had an unusually fulminant onset, with a poor prognosis suggested by previous criteria[3,4]. Vigorous treatment in this case has been worthwhile and at 6 months from onset active rehabilitation is continuing.

Diagnosis of GBS in the elderly may be overlooked because of its varied presentation and the possible coexistence of neurological disease. This illustration of how a very fulminant onset of disease may present with features suggestive of acute spinal cord compression shows that GBS can be an important, remedial cause of neurological disease in this age group, and how a positive attitude to treatment is justified.

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Meeting reports

Spongiform encephalopathies: implications of recent developments

Keywords: spongiform encephalopathies; man; animals

Report of joint meeting of Sections of Comparative Medicine, Epidemiology & Community Medicine and Pathology 15 November 1989

The spongiform encephalopathies are a group of diseases of man and animals which include Creutzfeldt-Jakob disease (CJD), Kuru, Gerstmann-Straussler-Scheinker disease, scrapie and transmissible mink encephalopathy. The most striking recent development in this field has been the emergence of a new disease; bovine spongiform encephalopathy (BSE). This new disease has captured the imagination of the press under headlines such as 'Mad Cow Disease' and 'Madness and Death in the Dairy Cow', and the possible implications of the condition for human and animal health have received wide attention. It was therefore no surprise that at the recent joint meeting of the sections of Comparative Medicine, Pathology, and Epidemiology & Community Health entitled 'Spongiform encephalopathies: implications of recent developments', BSE was the subject of five of the nine speakers.

Dr T Crowe (Northwick Park Hospital) began the meeting by discussing the spongiform encephalopathies of man, with particular reference to Gerstmann-Straussler-Scheinker disease (GSS). GSS is a rare disease which, unlike the sporadic spongiform encephalopathies of man, is transmitted as autosomal dominant familial cerebellar ataxia and dementia. Fifteen per cent of Creutzfeldt-Jakob disease cases also appear to be familial, but the GSS cases can be distinguished by their slower onset, the presence of cerebellar ataxia and by more prominent accumulation of amyloid in the brain. It has been known for some time that the protein found in the amyloid plaques is essential for transmission of spongiform encephalopathies and that it is coded for by the 'prion' gene. Detailed studies on a number of families with GSS and familial CJD, have identified several mutations in this gene. In three of the four mutations described the abnormal mutation was not seen in any unaffected individual, suggesting that it might be directly pathogenic. However, in the fourth example an alteration at codon 129 identified in a number of GSS families was also found in cases of familial CJD, sporadic CJD, iatrogenic (growth hormone therapy associated) CJD and in a proportion of normal individuals. This posed the intriguing question of whether there may be a susceptibility gene, similar to the SINC gene in mice, or indeed whether all the mutations so far described might in some way control susceptibility.

Dr J Hope (Neuropathogenesis Unit, Edinburgh) then reviewed the nature of the pathogenic agent. The infectious agent has long been known to be resistant to formalin and autoclaving. It is relatively insensitive to ionising radiation, although it does show a high oxygen enhancement ratio. This was previously thought to indicate that the infective particle consisted of lipid and polysaccharide, but is now considered to be consistent with it being a protein with an oxygen sensitive moiety. The molecular structure of the scrapie agent is still unknown but evidence has been accumulating in support of the hypothesis that although scrapie is caused by an environmental agent, prion protein (PRP) is involved. A possible role for PRP could be to act as a receptor site where the agent might attach and this kind of theory is now being tested using transgenic techniques. Recent experiments involved inserting the hamster PRP gene into mice. The mice did not develop scrapie but when challenged showed a greatly reduced incubation period.

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